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Remodeling muscles with calcineurin.

Olson EN, Williams RS.

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Ca(2+) signaling plays a central role in hypertrophic growth of cardiac and skeletal muscle in response to mechanical load and a variety of signals. However, the mechanisms whereby alterations in Ca(2+) in the cytoplasm activate the hypertrophic response and result in longterm changes in muscle gene expression are unclear. The Ca(2+), calmodulin-dependent protein phosphatase calcineurin has been proposed to control cardiac and skeletal muscle hypertrophy by acting as a Ca (2+) sensor that couples prolonged changes in Ca(2+) levels to reprogramming of muscle gene expression. Calcineurin also controls the contractile and metabolic properties of skeletal muscle by activating the slow muscle fiber-specific gene program, which is dependent on Ca(2+) signaling. Transcription factors of the NFAT and MEF2 families serve as endpoints for the signaling pathways whereby calcineurin controls muscle hypertrophy and fiber-type. We consider these findings in the context of a model for Ca(2+)-regulated gene expression in muscle cells and discuss potential implications of these findings for pharmacologic modification of cardiac and skeletal muscle function. BioEssays 22:510-519, 2000. Copyright 2000 John Wiley & Sons, Inc.

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